CDC
January 24, 1997 / Vol. 46 / No. 3

MMWR™

MORBIDITY AND MORTALITY WEEKLY REPORT

As part of its commemoration of CDC's 50th anniversary, MMWR *is reprinting selected* MMWR *articles of historical interest to public health, accompanied by a current editorial note.*

Reprinted below is the initial report published August 6, 1976, on an outbreak of respiratory illness among persons who attended an American Legion convention in Philadelphia during the summer of 1976. Following that report is the special issue of MMWR *published January 18, 1977, which announced the identification of the bacterium that caused Legionnaires disease. Tables from the special issue have been recreated to resemble the originals as closely as possible. A contemporary Editorial Note follows the outbreak reports.*

Epidemiologic Notes and Reports

Respiratory Infection — Pennsylvania

A total of 152 persons associated with a state American Legion convention in Philadelphia July 21–24 have been hospitalized with respiratory infections. Onsets of illness were in the period July 22–August 3; the majority occurred from July 25 to July 31. Twenty-two of these patients have died. The deaths, reported over the past week, were primarily due to pneumonia.

Although information about the disease and its epidemiology is incomplete, it appears to be characterized by the acute onset of fever, chills, headache, and malaise, followed by a dry cough and myalgia. Some of the most seriously ill developed high fever and died in shock with extensive pneumonia. No etiologic agent has yet been incriminated. There is no information available concerning other Legionnaires who may be ill with less severe symptoms.

The patients, among several thousand attending the convention, stayed in at least 3 or 4 hotels while in Philadelphia. There is no evidence of increase in respiratory disease in Philadelphia residents, nor has there been any confirmed secondary spread to family members or other contacts. There have been several reports of similar disease in non-conventioneers who were in Philadelphia at the same time as the convention.

Legionnaires Disease — Continued

Reported by RG Sharrar, MD, City of Philadelphia Dept of Public Health; WE Parkin, DVM, Acting State Epidemiologist, Pennsylvania State Dept of Health; the Bur of Epidemiology and the Bur of Laboratories, CDC.

[*From the January 18, 1977, special issue of* MMWR]

Epidemiologic Notes and Reports

Follow-up on Respiratory Illness — Philadelphia

Last summer an outbreak of severe respiratory illness occurred in Pennsylvania chiefly among those who had attended a state American Legion convention in Philadelphia July 21–24, 1976 (MMWR 25 [30,33,34]). An estimated 180 cases including 29 deaths occurred (MMWR 25 [38]). An organism has now been isolated in yolk sacs of embryonated hens' eggs that appears to be the etiologic agent. For the purpose of this report the yolk sac isolate is being called a bacterium on the basis of its size and morphology.

The bacterium was first isolated from the lung tissues of 1 fatal case of Philadelphia respiratory disease and 1 fatal case of Broad Street pneumonia (see below) by inoculation of guinea pigs intraperitoneally. After a 1- to 2-day incubation period the guinea pigs developed a febrile illness that was characterized in most animals by watery eyes and prostration. Spleen suspensions of febrile guinea pigs were inoculated into yolk sacs of embryonated eggs from antibiotic-free chicken flocks. The embryos died after 4–6 days, and Gimenez-stained smears of the yolk sacs were found by microscopic examination to contain many bacilli. The bacilli were gram-negative and moderately pleomorphic. Surviving guinea pigs were shown by indirect immunofluorescence to have developed antibody to the yolk sac isolates. Because most bacteria when inoculated into the yolk sac kill the eggs in 1–2 days, an unusual rickettsia was suspected. The organism is bigger than a rickettsia, however, and the convalescent guinea pig sera failed to react in the complement fixation test with standard rickettsial antigens prepared from *Coxiella burnetii, Rickettsia rickettsii, R. prowazekii,* and *R. typhi.* Cultivation on sheep blood agar and Trypticase Soy Agar has been attempted at each yolk sac passage. Frequently, no growth has been observed, but yolk sacs infected with 1 isolate have sometimes given many minute colonies after 2–3 days' incubation. The slowness of growth has delayed bacteriological identification.

Evidence for the etiologic role of the yolk sac isolate in the epidemic has been obtained by indirect fluorescent antibody stains carried out by methods that are the same as those in regular use in the diagnosis of rickettsial diseases, except that the microdrops fixed to the slide were prepared from yolk sacs infected with isolate 1 and isolate 2 of the Philadelphia agent. The results in Tables 1 and 2 were obtained with sera from 33 patients who were selected because they were Legionnaire delegates who were hospitalized, survived, and had radiologic evidence of pneumonia and fevers of at least 102 F; they thus represented the most typical survivors.

Table 1 shows some representative results. The sera with high titers gave bright staining at low dilutions which gradually decreased with increasing dilution. The brightness of staining and height of the titers are similar to that observed in other infectious diseases, for example, Rocky Mountain spotted fever. Patients 1, 2, and

Legionnaires Disease — Continued

TABLE 1. Results with indirect fluorescent antibody stains of the agent cultivated in yolk sacs. Sera from selected patients with Philadelphia respiratory disease.

Patient	Specimen	Day of Disease	16[a]	32	64	128	256	512	Titer	Interpretation[b]
1	S1	1	1+/1+[c]	±/±	0/0	0/0	0/0	0/0	16/16[d]	
	S2	22	3+/3+	3+/3+	2+/3+	1+/2+	±/1+	0/0	128/256	C
2	S1	4	±/±	0/0	0/0	0/0	0/0	0/0	<16/<16	
	S2	11	2+/3+	2+/2+	1+/2+	1+/1+	±/±	0/0	128/128	
	S3	25	3+/3+	2+/2+	2+/2+	1+/1+	±/1+	±/±	128/256	C
3	S1	24	±/±	±/±	0/0	0/0	0/0	0/0	<16/<16	
	S2	34	3+/3+	3+/3+	2+/3+	2+/3+	1+/2+	1+/1+	>512/>512	C
4	S2	12	3+/3+	2+/2+	1+/1+	±/±	0/0	0/0	64/64	
	S3	33	3+/3+	2+/2+	1+/2+	1+/1+	±/1+	0/0	128/256	C
5	S1	8	2+/2+	2+/2+	1+/1+	1+/1+	±/±	0/0	128/128	
	S3	29	3+/3+	3+/3+	2+/3+	2+/±	±/0	0/0	128/64	P
6	S1	5	0/0	0/0	0/0	0/0	0/0	0/0	<16/<16	
	S3	29	0/0	0/0	0/0	0/0	0/0	0/0	<16/<16	N

[a] The reciprocal of the dilution is shown in this and other tables.

[b] C = seroconversion or increase in titer of at least 4-fold with 1 or both antigens. P = classified as positive because the titer was high but showed little change. N = classified as negative because all specimens had low titers.

[c] Brightness of staining: 0 = no staining, ± = questionable staining, 1+ = barely detectable but definite staining, 2+ and 3+ = increasing brightness. The 2 numbers refer to staining of the bacteria in yolk sacs infected with the first 2 isolates.

[d] Highest dilution with definite staining with either antigen.

TABLE 2. Results with indirect fluorescent antibody stains of the agent cultivated in yolk sacs: Summary of results with patients with Philadelphia respiratory disease

Interpretation of Titers		Number of Patients
Seroconversions: >4-fold		19
4-fold		5
Positive (≥64) without seroconversion		5
Negative		4
Maximum titer observed with seroconversions and positives	8192	1
	2048	1
	1024	3
	>512	3
	512	6
	256	6
	128	6
	64	3
Negatives	<64	4
Total patients tested		33

Legionnaires Disease — Continued

3 had distinct increases in antibody titers, and 4 had only a 4-fold increase. The first specimen from patient 5 was already at a titer of 128, and there was no further increase between the eighth and twenty-ninth day of illness.

Table 2 summarizes the results with sera of the 33 Legionnaire patients tested to date; 29 gave results that suggest they were infected with the organism. Seroconversions were seen in 25 patients and antibody rises of more than 4-fold in 19. The maximum titers observed were 128 or greater in 26 out of 29 patients. The titers were usually low in the first week of illness, but they rose rapidly in the second and third weeks. The fact that 3 patients had no serologic response is not surprising since the cases were defined on a clinical and epidemiological basis. The staining of isolates 1 and 2 has been very similar with these sera and with the other sera reported below. Thus the 2 yolk-sac isolates are antigenically very similar if not identical.

Cases of Broad Street pneumonia represent disease clinically similar to Philadelphia respiratory disease that occurred in persons who did not attend the Convention, were within 1 block of Hotel A between July 1–August 18, but said they did not go into Hotel A during the epidemic period. Sera from 4 of the 38 such patients have been tested. Two have shown serologic conversions from titers of 16 or less to 512 or greater. Two had unchanging titers of 32 or less.

As controls for the fluorescent antibody tests, sera were tested from 40 patients unrelated to the outbreak whose specimens had been submitted for rickettsial diagnosis (Table 3). The rickettsial complement fixation tests had failed to demonstrate rickettsial antibody. The sera were first screened at a dilution of 1:32 and those with staining at this dilution were retested at dilutions of 1:16 through 1:512. Most of the titers observed with the yolk sac isolate were low. Two specimens had titers of 64, that is, they overlapped with the lowest titers observed in Legionnaire patients in Table 2. The staining at low dilutions with these sera was only 1+ bright; however, in all the seropositive Legionnaire patients in Table 2 and in the 2 Broad Street pneumonia patients who converted, fluorescence was 3–4+ bright in low dilutions.

TABLE 3. Results with sera from control patients clinically suspected to have Rickettsia infections

Titer	Number of Persons
64	2
32	6
16	3
<32	28
<16	1
TOTAL	40

TABLE 4. Serologic results with other persons who did not meet clinical criteria for a case of Philadelphia respiratory disease[a]

	Titers						
	<16	16	32	64	128	256	Total
Hotel employee, lobby		1	2				3
Hotel employee, non-lobby	6	4	2			1	13
Legionnaire, not at convention	3	5	1	2			11

[a] In all these sera the staining, even at low dilutions, was not more than 1+ (barely detectable).

Legionnaires Disease — Continued

In the outbreak, illness in conventioneers was associated with time spent in Hotel A. The incidence was directly related to time spent in the lobby. Sera were available from some hotel employees of 2 categories: those who worked in the lobby and those who worked in locations removed from the lobby (Table 4). Also shown in the table are the results with sera from a group of Pennsylvania Legionnaires who did not attend the convention. One positive titer in a hotel employee was seen, a cashier checker, who had a titer of 256. The titers with the other employees and the Legionnaires who did not attend the convention were within the range of the 40 non-epidemic sera reported in Table 3.

In 1966 an outbreak of acute pneumonia occurred at a large psychiatric hospital in the District of Columbia. There were 94 cases and 16 deaths. Acute and convalescent sera were available from 14 patients; they were also tested against the antigens from Isolate 1 and 2. Thirteen had distinct rises in titer of 8-fold or more, and 12 had titers of 128 or more. The brightness of staining and titers were the same as those seen with the Legionnaire patients.

The intensity of public interest in the Philadelphia epidemic makes it necessary to provide a factual account of these findings now. The etiology of the outbreak has been unknown. The present findings provide very strong evidence that the 2 epidemics were caused by the bacterium isolated in yolk sacs and that nearly all the cases had the same cause. The bacterium can be identified now by the characteristic disease it produces in guinea pigs, the characteristic death pattern in eggs, the at best dysgonic growth on the bacterial media tried, and by the fluorescent antibody staining results. Other, more complex explanations are possible. For example, the bacterium might be thought of as a secondary invader associated with a virus, but extensive virological searches have failed to reveal a virus and the serologic responses for the bacterium have been present in a very high percentage of the cases. There has not been time to identify the organism taxonomically. The source of the organism in the outbreak is not known, but the search should now be greatly facilitated.

Reported by the Leprosy and Rickettsia Br, Virology Div, Bur of Laboratories, CDC.

Follow-up Survey Data: In December 1976, selected survivors of Philadelphia respiratory disease and matched controls were interviewed concerning smoking habits, liquor and snack food preference, and knowledge of homemade liquor. The 56 patients selected for interview represented all hospitalized male survivors who had been delegates to the American Legion convention and were known to have developed an illness characterized by temperature of 102 F or higher and pneumonia proved by X-ray. The 56 controls were male delegates matched by age who had indicated on earlier survey that they had not been ill since the convention. The interviews were completed with 52 case-control pairs. Cigarette smoking habits at the time of the convention were the only significant associations with illness. The relative risk of illness

TABLE 5. History of cigarette smoking at the American Legion Convention, Philadelphia, July 1976 among case-control pairs

	Cases		
Controls	Smoker	Non-smoker	Total
Smoker	14	5	19
Non-smoker	17	16	33
Total	31	21	52

Legionnaires Disease — Continued

among cigarette smokers was 3.4 compared to non-smokers ($X^2(1) = 5.5$, $p < .05$, McNemar) (Table 5). Cases also smoked more cigarettes and were more likely to have smoked sample cigarettes available at the convention. A previous survey showed no single cigarette brand common among cases. Pipe or cigar smoking was not associated with illness.

Reported by RG Sharrar, MD, City of Philadelphia Dept of Public Health; E Streiff, RN, MPH, Allegheny County Dept of Health; WE Parkin, DVM, Acting State Epidemiologist, Pennsylvania State Dept of Health; Bur of Epidemiology and Bur of Laboratories, CDC.

Editorial Note—1997: A bacterial etiology was not initially evident during the field investigation phase of the Legionnaires disease outbreak. Chest radiographs of case-patients revealed an interstitial pneumonia, which at that time was considered indicative of a viral infection. Because the Legionnaires disease bacterium is refractory to most stains, no bacteria could be visualized when sections of lung tissues from deceased patients were stained by commonly used methods, such as the Brown-Brenn technique. Additionally, no species of bacteria was reproducibly isolated from autopsy materials or clinical specimens because the special nutritional requirements of the Legionnaires disease bacterium precluded its growth on conventional culture media. The bacterium was, however, isolated in guinea pigs and in the yolk sacs of embryonated hens' eggs and was visualized by Gimenez stain during one of several efforts to isolate Q fever rickettsiae (*Coxiella burnetii*) from specimens of lung tissue collected at autopsy. It subsequently was cultivated on enriched Mueller-Hinton agar using heavily infected yolk sacs as inoculum. The unique nutritional requirements of the bacterium were identified in separate studies, and a new culture medium was developed that now allows routine isolation of the Legionnaires disease bacterium from clinical specimens (*1*).

Determination of the phenotypic and genotypic properties of the Philadelphia isolate indicated that it was a novel species (*Legionella pneumophila*) (*2*). The genus *Legionella* now comprises approximately 40 named species and subspecies that are associated with water. Approximately half of the species have been implicated in human disease; *L. pneumophila* serotype 1, the prototype strain that was isolated following the Philadelphia outbreak, is responsible for most infections.

The epidemic of pneumonia that followed the American Legion convention in August 1976 was one of the most publicized epidemics in which CDC had participated. Daily newspaper reports contained "body counts," rumors of biological and chemical warfare, and accusations of cover-up by CDC. Reports by CDC in the *MMWR*, however, were limited to short back-page accounts, reporting that the epidemic had occurred and was under investigation.

On Friday, January 14, 1977, the director of CDC's Laboratory Division, Charles Shepard, M.D., and microbiologist Joseph E. McDade, Ph.D., went to the office of the CDC Director, David J. Sencer, M.D. After a few hesitant moments, they informed him that they had isolated the agent that had caused the outbreak. Dr. Shepard wanted to take the weekend to redo the isolation in a laboratory where they had not been working to rule out any possibility of contamination.

Although Dr. Shepard did not want to release the information until it was published in the peer-reviewed scientific literature, Dr. Sencer wanted to fulfill CDC's responsibility to immediately release the information to state and local health departments because the outbreak had been in the national news for months and this

Legionnaires Disease — Continued

information could prevent other cases. A solution was found: *MMWR* is a scientific publication, and CDC published and printed *MMWR*. CDC could print a special edition on the following Tuesday, January 18, 1977 (normal publication was on Thursdays). Once it was in print at 1 p.m., it could be given to the news media with an embargo until 3 p.m.

Tuesday morning, as the presses were beginning to run 180,000 copies, Dr. Shepard reported that he had retrieved serum specimens from the serum bank of two earlier unsolved outbreaks of pneumonia, and they were positive for the identical organism. The presses were stopped, and the changes were made.

At 1 p.m., a conference call was scheduled from CDC to the state health officers, the Surgeon General, the National Institutes of Health, and other public health officials participating in the investigation. CDC employees who had worked in any way on Legionnaires disease—from dishwashers in the laboratory to the chiefs of epidemiology and the laboratory—were invited to this conference call. Following that call, CDC conducted a press conference, in which Drs. Shepard and McDade presented the findings and distributed the *MMWR*. This is the only occasion on which an extra issue of the *MMWR* (weekly) has been published.

Legionnaires disease is only one of many new diseases, syndromes, or etiologic agents that have been identified during the past 2 decades (*3*), and CDC has responded to these new challenges. Other noteworthy examples include Lyme disease, human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS), toxic-shock syndrome, human ehrlichiosis, hantavirus pulmonary syndrome, hepatitis C virus, and *Escherichia coli* O157. In some instances (for example, HIV/AIDS), both the disease and its etiologic agent previously were unknown to medical science. In others, the disease already existed but was unrecognized. Legionnaires disease apparently occurred sporadically as early as the 1940s. Retrospective analysis of a "rickettsia-like agent," which was isolated from a pneumonia patient in 1947, revealed that this agent was identical to *L. pneumophila* (*4*). However, at the time of its isolation, the source of the bacterium was erroneously attributed to the guinea pigs that were used in the isolation procedure. Since the 1940s, other technologic changes, including the introduction of air-conditioning cooling towers, have facilitated the potential for exposure through dissemination of infectious aerosols of *Legionella* in contaminated water (*5*). This realization has lead to changes in routine maintenance procedures for many aerosol-producing devices such as cooling towers, spas, and respiratory therapy equipment. The development of prevention strategies is an ongoing process involving medical professionals, engineers, and chemical disinfectant manufacturers.

Identification of new etiologic agents undoubtedly will continue. For example, of the approximately 4 million cases of pneumonia that occur in the United States each year, the etiologic agent remains unidentified in up to 50% of cases even when an etiology is actively sought (*6*). Similarly, no etiologic agent is found in 60% of reported foodborne disease outbreaks, most of which are studied using routine diagnostic methods, nor in 32% of diarrheal outbreaks on cruise ships, despite intensive investigation (*7,8*). However, in contrast to the Legionnaires disease investigation, during which the etiologic agent was identified serendipitously, new molecular techniques allow for a more systematic search for infectious etiologies. In particular, the extreme sensitivity of representational difference analysis and consensus sequence-based

Legionnaires Disease — Continued

polymerase chain reaction technology should allow the identification of many etiologic agents that previously have been refractory to culture (*9*).

Editorial Note by: David J Sencer, MD, former Director, Center for Disease Control. Joseph E McDade, PhD, Associate Director for Laboratory Science, National Center for Infectious Diseases, CDC.

References

1. Feeley J, Gibson RJ, Gorman GW, et al. Charcoal-yeast extract agar: primary isolation medium for *Legionella pneumophila*. J Clin Microbiol 1979;10:437–41.
2. Brenner DJ, Steigerwalt AG, McDade JE. Classification of the Legionnaires' disease bacterium: *Legionella pneumophila*, genus novum, species nova, of the family *Legionellaceae*, familia nova. Ann Intern Med 1979;90:656–8.
3. Institute of Medicine. Emerging infections: microbial threats to health in the United States. Washington, DC: National Academy Press, 1992.
4. McDade JE, Brenner DJ, Bozeman FM. Legionnaires' disease bacterium isolated in 1947. Ann Intern Med 1979;90:659–61.
5. Breiman RF. Impact of technology on the emergence of infectious diseases. Epidem Rev 1996;18:4–9.
6. Marston BJ. Epidemiology of community-acquired pneumonia. Infectious Diseases in Clinical Practice 1995;4(suppl 4):S232–S239.
7. Bean NH, Goulding JS, Lao C, Angulo FJ. Surveillance for foodborne-disease outbreaks—United States, 1988–1992. In: CDC surveillance summaries (October). MMWR 1996;45 (no. SS-5).
8. Koo D, Maloney K, Tauxe R. Epidemiology of diarrheal disease outbreaks on cruise ships, 1986 through 1993. JAMA 1996;275:545–7.
9. Gao SJ, Moore PS. Molecular approaches to the identification of unculturable infectious agents. Emerging Infectious Diseases 1996;2:159–67.

Prevalence of Cigarette Smoking Among Secondary School Students — Budapest, Hungary, 1995

Because of the high prevalence of tobacco use in countries of Central and Eastern Europe, public health officials in many of these countries have designated as a priority the prevention of smoking initiation among youth. In 1995, a nationally representative survey in the Republic of Hungary documented that 35.8% of 16-year-old students in that country had smoked cigarettes during the preceding 30 days (*1*). To better characterize smoking among youth in Hungary, the Field Epidemiology Training Program, Hungarian Ministry of Welfare, conducted a cross-sectional survey in Budapest (1995 population: 1,906,798) among secondary school students aged 14–18 years. Specific objectives of the survey were to assess the prevalence of cigarette smoking among these students, determine factors associated with higher prevalences, and describe the smoking habits of current cigarette smokers. This report summarizes the findings, which indicate that one third of all students smoked; half of all 18-year-olds smoked; and of those students who smoked, 41% most frequently smoked an imported, internationally recognized cigarette brand.

Among the 105,209 Budapest students aged 14–18 years, approximately 80% attended traditional public high schools, and 20% attended public vocational/technical schools. A sample of students was selected from a stratified sample of the 199 secondary schools in Budapest. Twenty (80%) traditional high schools and five

Cigarette Smoking — Continued

(20%) vocational/technical schools were selected with a probability proportional to their size. Classrooms in these 25 schools were then randomly selected. During 3 weeks in January 1995, all 2878 students in attendance completed a pretested, standardized questionnaire that included questions translated from the U.S. Youth Risk Behavior Survey (*2*) and that asked about culturally relevant factors possibly associated with smoking. Current smokers were defined as students who reported having smoked at least one cigarette during the preceding 30 days. Of the 2878 students, 79 (2.7%) were excluded because their smoking status could not be determined. Epi Info 6.02 was used for data analysis that accounted for the stratification and clustering of students within classrooms; 95% confidence intervals (CIs) were calculated using SUDAAN (*3*).

Among the 2799 students, 987 (35.3%) (95% CI=30.6%–39.9%) reported current smoking (Table 1). Although the prevalences were similar among male and female students (prevalence odds ratio [POR]=1.0; 95% CI=0.8–1.5), students aged 18 years were more likely to smoke than students aged 14 years (47.9% and 23.8%, respectively [POR=2.9; 95% CI=1.3–6.6]). The prevalences of current smoking also were higher among vocational/technical students than traditional high school students (53.1% and 31.0%, respectively [POR=2.5; 95% CI=1.6–3.9]); among students whose friends smoked than those whose friends did not smoke (42.6% and 6.8%, respectively [POR=10.1; 95% CI=7.5–13.7]); among students who reported that they had seen a teacher smoking during the school year than those who had not seen a teacher smoking (37.3% and 19.0%, respectively [POR=2.5; 95% CI=1.8–3.6]); and among students with a family member who smoked than students whose family members abstained from smoking (40.7% and 27.0%, respectively [POR=1.9; 95% CI=1.6–2.1]). The prevalences of smoking were similar among students who received instruction at

TABLE 1. Number and percentage of current smokers* among secondary school students aged 14–18 years, by selected characteristics — Budapest, Hungary, 1995

Characteristic	Sample size†	Current smokers		
		No.	(%)	(95% CI§)
Sex				
Male	1470	525	(35.7)	(28.5%–42.9%)
Female	1324	461	(34.8)	(32.2%–37.5%)
Age (yrs)				
14	168	40	(23.8)	(23.0%–24.7%)
15	720	191	(26.5)	(20.5%–32.6%)
16	806	286	(35.5)	(27.6%–43.4%)
17	696	274	(39.4)	(34.6%–44.2%)
18	399	191	(47.9)	(32.9%–62.9%)
School type				
Vocational/ technical	537	285	(53.1)	(46.7%–59.5%)
Traditional high school	2262	702	(31.0)	(26.2%–35.9%)
Total	**2799**	**987**	**(35.3)**	**(30.6%–39.9%)**

*Defined as students who reported having smoked at least one cigarette during the preceding 30 days.
†For some characteristics, the sample size may not equal 2799 because of missing data.
§Confidence interval.

Cigarette Smoking — Continued

school about the harmful health effects of smoking and among those who did not receive such instruction (POR=1.0; 95% CI=0.9–1.1).

Among current smokers, during the preceding 30 days, 17.3% smoked ≥11 cigarettes daily, 38.0% smoked daily, and approximately half (51.0%) smoked on school property on at least 1 day (Table 2). Approximately 60% of current smokers smoked a variety of brands of cigarettes. Current smokers reported that the brands they most frequently smoked were Hungarian brands (Multifilter [57%] and Sopianae [33%]) and a U.S. brand (Marlboro [41%]).

Reported by: G Ursicz, MD, Hungarian Field Epidemiology Training Program, Ministry of Welfare; É Kiss, MD, Div of Child and Adolescent Health, K Lun, MD, Director, Budapest Institute of Public Health and Medical Officer Svc, Ministry of Welfare; Ministry of Culture and Education, Budapest, Republic of Hungary. Div of International Health (proposed), Epidemiology Program Office; Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion, CDC.

Editorial Note: The findings of the survey described in this report indicate that in 1995, a substantial proportion (35%) of secondary school students in Budapest reported smoking cigarettes. This prevalence is identical to that among U.S. students in grades 9–12 during 1995 (*2*); however, the findings for the United States reflected a national sample of persons who resided in urban and rural areas, and the findings for Hungary reflected a sample of persons who resided in one large urban area. The prevalence of smoking in Budapest increased directly with age and was 48% among 18-year-old students. Worldwide, about half of persons who initiate smoking during their teenage

TABLE 2. Number and percentage of secondary school students aged 14–18 years who were current smokers*, by selected characteristics — Budapest, Hungary, 1995†

Characteristic	Current smokers		
	No.§	(%)	(95% CI¶)
No. cigarettes smoked per day			
1	223	(23.3)	(20.3%–26.2%)
2–10	569	(59.4)	(57.3%–61.5%)
≥11	166	(17.3)	(14.1%–20.5%)
No. days used			
1– 2	201	(20.4)	(17.3%–23.5%)
3– 9	148	(15.0)	(12.1%–17.9%)
10–29	263	(26.6)	(23.2%–30.1%)
30	375	(38.0)	(34.7%–41.3%)
No. days used on school property			
0	469	(49.0)	(42.6%–55.3%)
1– 2	98	(10.2)	(7.5%–13.0%)
3– 9	109	(11.4)	(8.6%–14.2%)
≥10	282	(29.4)	(24.7%–34.2%)

*Defined as students who reported having smoked at least one cigarette during the preceding 30 days.
†n=987.
§For each characteristic, the sample size does not equal 987 because of missing data.
¶Confidence interval.

Cigarette Smoking — Continued

years and continue to smoke cigarettes regularly will die as a result of a tobacco-related disease (*4*). The death rates for diseases attributable to smoking are higher in Hungary than in most other developed countries (*4,5*).

A survey of the prevalence of smoking among adolescents in European countries during 1993–1994 indicated that among five countries in central and eastern regions (Czech Republic, Hungary, Republic of Poland, Russian Federation, and Slovak Republic), approximately 10% of adolescents reported smoking cigarettes at least weekly. However, the overall prevalence of cigarette smoking for all age groups in Hungary is among the highest of all countries in central and eastern Europe. Each year from 1976 through 1990, annual average per capita cigarette consumption in Hungary was higher than the combined average for all central and eastern European countries (*5*).

The finding that most current smokers varied the brand of cigarette they smoked may reflect the ease with which students can purchase individual cigarettes at newsstands and other stores in Hungary. Students may vary the brand of cigarette they smoke based on the availability and cost of individual cigarettes. In general, in Budapest, imported western brand-name cigarettes are more expensive than central and eastern European brand-name cigarettes.

To decrease the initiation and prevalence of smoking in Hungary, health officials are developing a population-based tobacco education campaign that will include a pre- and postintervention smoking prevalence survey to evaluate the impact of the program. In addition, a pilot intervention project is being planned in a large city (Szekesfehérvár) to decrease exposure to passive smoke (environmental tobacco smoke); this project will include both a general media campaign and a program to educate kindergarten children and their parents about the hazards of passive and active smoking. Public health officials in Budapest also have recommended that teachers who smoke do so in restricted areas that are out of sight of students.

Although cigarette advertising that actively promotes the purchase of cigarettes is prohibited in Hungary, such advertising is common in many public locations, including sports arenas, large city squares, housing complexes, and busy traffic intersections. Public health officials also have recommended stronger enforcement of the ban on cigarette advertising (E. Morava, Hungarian Ministry of Welfare, personal communication, 1996).

References

1. Elekes Z, Paksi B. The European School Survey Project on Alcohol and Drugs [Hungarian]. Budapest, Hungary: Hungarian Ministry of Welfare, 1996.
2. CDC. Tobacco use and usual source of cigarettes among high school students—United States, 1995. MMWR 1996;45:413–8.
3. Shah BV. Professional software for Survey Data Analysis (SUDAAN), version 6.40 [Software documentation]. Research Triangle Park, North Carolina: Research Triangle Park Institute, 1995.
4. Peto R, Lopez AD, Boreham J, Thun M, Health C Jr. Mortality from tobacco in developed countries: indirect estimation from national vital statistics. Lancet 1992;339:1268–78.
5. World Health Organization. Health for all [Software documentation]. Geneva, Switzerland: World Health Organization, 1995.

Outbreaks of Pneumococcal Pneumonia Among Unvaccinated Residents in Chronic-Care Facilities — Massachusetts, October 1995, Oklahoma, February 1996, and Maryland, May–June 1996

During October 1995–June 1996, CDC and state and local public health agencies investigated outbreaks of pneumococcal pneumonia with bacteremia at chronic-care facilities (CCFs) serving predominantly elderly populations in Massachusetts, Oklahoma, and Maryland. This report summarizes these investigations and identifies measures that may prevent such outbreaks.

In the investigation of these outbreaks, pneumonia was defined as a chest radiograph consistent with pneumonia and compatible clinical features. Control measures implemented in these outbreaks included restricting group activities and new admissions to the CCFs, cohorting of staff-resident assignments, and vaccination of all residents lacking documentation of pneumococcal polysaccharide vaccine (PV). In two of the outbreaks, antimicrobial prophylaxis was administered in conjunction with PV, after which no additional cases of invasive pneumococcal infection were identified.

Massachusetts. During October 5–14, 1995, a total of 10 cases of pneumonia occurred among 67 residents of a CCF (attack rate [AR]=14.9%); two cases were fatal. The median age of the residents was 90 years. *Streptococcus pneumoniae* serotype 14 was isolated from a blood culture obtained from one patient, and *S. pneumoniae* infection was confirmed by polymerase chain reaction during postmortem examination of lung tissue of another resident. Only three (4.5%) residents had documentation of vaccination with PV before the outbreak.

Oklahoma. During February 6–20, 1996, of 80 elderly residents at a CCF, 12 (15%) were hospitalized with pneumonia, and three residents with bacteremia died. The median age of residents was 85 years. Multidrug-resistant *S. pneumoniae* serotype 23F was isolated from blood cultures obtained from four residents and from sputum cultures from two others. The strain was resistant to penicillin, cefotaxime, cefaclor, chloramphenicol, erythromycin, trimethoprim-sulfamethoxazole, tetracycline, and clindamycin. *S. pneumoniae* serotype 23F with the same pattern of antimicrobial susceptibility also was isolated from the nasopharynx of 17 (23%) of 74 asymptomatic residents and two (3%) of 69 asymptomatic employees. Only three (3.8%) residents had documentation of previous vaccination with PV.

Maryland. During May 26–June 17, 1996, a total of 14 cases of pneumonia were identified among residents at a CCF with a census of 120 (AR=11.7%). The median age of patients was 86 years. *S. pneumoniae* serotype 4 was isolated from the blood of four residents with pneumonia. Four residents with pneumonia died, three of whom had pneumococcal bacteremia. Isolates were susceptible to the antimicrobials tested (penicillin, cefotaxime, chloramphenicol, erythromycin, trimethoprim-sulfamethoxazole, tetracycline, clindamycin, rifampin, ofloxacin, and vancomycin). Only two (1.9%) residents had documentation of previous vaccination with PV.

Reported by: P Kludt, MPH, SM Lett, MD, A DeMaria, Jr, MD, State Epidemiologist, Massachusetts Dept of Public Health. JM Crutcher, MD, State Epidemiologist, Oklahoma State Dept of Health. NM Curtis, CW Garvey, MD, LL Frank, Montgomery County Dept of Health and Human Svcs; A Danner, MPH, GC Benjamin, MD, D Dwyer, MD, State Epidemiologist, Maryland Dept of Health and Mental Hygiene. Adult Vaccine Preventable Diseases Br, National Immunization Program; Childhood and Respiratory Diseases Br, Div of Bacterial and Mycotic Diseases, National Center for Infectious Diseases, CDC.

Pneumococcal Pneumonia — Continued

Editorial Note: Infectious diseases are the third leading cause of death in the United States, and respiratory infections account for nearly one half of these deaths (*1*). During 1980–1992, pneumonia accounted for 85% of fatal respiratory infections among persons aged ≥65 years (*1*). *S. pneumoniae* is the most common cause of nursing-home–acquired pneumonia (*2*). Although PV is safe, efficacious, and cost-effective in reducing the incidence of bacteremic pneumococcal illness (*3*), only 30% of persons aged ≥65 years have been vaccinated (*4*).

Positive blood cultures are found in 20% of persons hospitalized with cases of pneumonia attributed to *S. pneumoniae* (*5*). This, along with the common practice of treating pneumonia empirically without first obtaining cultures, contributes to the disparity between the number of clinical cases of pneumonia and the number of bacteriologically confirmed cases described in this report.

The death rate among those CCF residents with pneumonia described in this report ranged from 20% to 28%, and <5% of the residents aged ≥65 years had documentation of previous vaccination with PV. The serotypes associated with disease in each outbreak are included in the 23-valent PV. Advisory Committee on Immunization Practices (ACIP) guidelines (*6*) recommend that all persons aged ≥65 years or others at increased risk for pneumococcal disease receive PV. Although most residents at the CCFs involved in this report were eligible for vaccination based on their age, documented vaccine coverage at these CCFs ranged from 1.9% to 4.5%, substantially below national estimates for persons aged ≥65 years and the national health objectives for the year 2000 (80%) (objective 20.11) (*7*). The recent emergence of antimicrobial resistance among *S. pneumoniae* (*8*) underscores the importance of PV use in accordance with ACIP guidelines, particularly among the institutionalized elderly.

At least two factors contribute to the low rate of PV vaccination among the institutionalized elderly. First, physicians for residents in CCFs usually do not emphasize administration of PV (*9*). Second, incomplete documentation of vaccination history for CCF residents and misconceptions about adverse reactions after unintended revaccination with PV may discourage health-care providers from vaccinating CCF residents with unknown vaccination history; however, the incidence of serious adverse events is as low following revaccination as it is following initial vaccination (*10*).

The risk for outbreaks of invasive pneumococcal infection in CCFs can be reduced by ensuring that all residents aged ≥65 years have been offered PV. During outbreaks of respiratory illness in CCFs, febrile residents should have cultures conducted before starting antimicrobial therapy. New admissions to CCFs should be encouraged to receive PV before admission to the facility or be offered vaccination on admission. To decrease the administration of unnecessary doses of PV, vaccination status should be recorded for each resident. When eligible residents are uncertain about their vaccination history or the resident's medical record is incomplete, vaccine should be administered. PV can be administered simultaneously with influenza vaccine; during annual influenza vaccine campaigns at CCFs, records should be reviewed to verify documentation of PV, and PV should be offered to all residents lacking documentation. Administrative and legislative policies also may assist in requiring CCFs and other health-care institutions to offer PV and other vaccines to eligible adults.

References

1. Pinner RW, Teutsch SM, Simonsen L, et al. Trends in infectious diseases mortality in the United States. JAMA 1996;275:189–93.

Pneumococcal Pneumonia — Continued

2. Marrie TJ, Slayter KL. Nursing home-acquired pneumonia: treatment options. Drugs Aging 1996;8:339–48.
3. Butler JC, Breiman RF, Campbell JF, Lipman HB, Broome CV, Facklam RR. Pneumococcal polysaccharide vaccine efficacy: an evaluation of current recommendations. JAMA 1993; 270:1826–31.
4. CDC. Influenza and pneumococcal vaccination levels among adults aged ≥65 years—United States, 1993. MMWR 1996;45:853–9.
5. Musher DM. Pneumococcal pneumonia including diagnosis and therapy of infection caused by penicillin-resistant strains. Infect Dis Clin North Am 1991;5:509–21.
6. CDC. Pneumococcal polysaccharide vaccine: recommendations of the Immunization Practices Advisory Committee. MMWR 1989;38:64–8,73–6.
7. Public Health Service. Health people 2000 review, 1995–96. Washington, DC: US Department of Health and Human Services, Public Health Service, 1996.
8. Butler JC, Hofmann J, Cetron MS, et al. The continued emergence of drug-resistant *Streptococcus pneumoniae* in the United States: an update from the Centers for Disease Control and Prevention's Pneumococcal Sentinel Surveillance System. J Infect Dis 1996;174:986–93.
9. Quick RE, Hoge CW, Hamilton DJ, Whitney CJ, Borges M, Kobayashi JM. Underutilization of pneumococcal vaccine in nursing homes in Washington State: report of a serotype-specific outbreak and a survey. Am J Med 1993;94:149–52.
10. Snow R, Babish JD, McBean AM. Is there any connection between a second pneumonia shot and hospitalization among Medicare beneficiaries? Public Health Reports 1995;110:720–5.

Antibiotic Resistance Among Nasopharyngeal Isolates of *Streptococcus pneumoniae* and *Haemophilus influenzae* — Bangui, Central African Republic, 1995

Approximately 4 million children aged <5 years die worldwide each year from acute respiratory infections (ARI), most of which are pneumonia (*1*). Most pneumonia deaths result from bacterial infections, and *Streptococcus pneumoniae* (SP) and *Haemophilus influenzae* (HI) are the most common bacterial etiologies (*1*). To provide data about antibiotic resistance and to assist the National ARI Control Program of the Central African Republic (CAR) (1995 population: 2.9 million) in choosing which antibiotics to recommend for the treatment of pneumonia in children aged <5 years, a survey of the antibiotic resistance of nasopharyngeal (NP) isolates of SP and HI cultured from children residing in Bangui (1995 population: 451,000), CAR, was conducted during January 16–February 8, 1995, by the Ministry of Public Health and Population (MOPHP) in collaboration with epidemiologists from CDC and microbiologists from the South African Institute for Medical Research. Bangui is the capital of and the largest city in CAR. The decision to measure resistance rates among NP isolates was based on the results of a study indicating that resistance rates of SP and HI isolates cultured from NP swabs were similar to rates measured among isolates cultured from blood (*2*). This report summarizes the results of that survey, which indicated that SP and HI had relatively low resistance rates to penicillin, ampicillin, cotrimoxazole (trimethoprim-sulfamethoxazole), and chloramphenicol.

Specimens for culture were obtained from 371 consecutive children who received consultation for any illness at two outpatient clinics in Bangui. In addition, to determine resistance rates in a population at higher risk for carriage of a resistant organism, specimens were obtained from 35 children hospitalized on an inpatient ward. NP cultures of the 371 outpatients yielded 272 SP isolates and 73 HI isolates. NP cultures

S. pneumoniae *and* H. influenzae — *Continued*

TABLE 1. Number and percentage of antibiotic-resistant* nasopharyngeal isolates of *Streptococcus pneumoniae* and *Haemophilus influenzae* cultured from children aged 2–59 months who were either inpatients or outpatients, by resistance level — Bangui, Central African Republic, January–February, 1995

Organism/ Antibiotic	Inpatients				Outpatients			
	Intermediate	High	Total		Intermediate	High	Total	
	No.	No.	No.	(%)	No.	No.	No.	(%)
S. pneumoniae	n=17 isolates				n=272 isolates			
Penicillin	5	0	5	(29.4)	24	0	24	(8.8)
Cotrimoxazole†	4	0	4	(23.5)	15	2	17	(6.3)
Tetracycline	1	10	11	(64.7)	17	98	115	(42.3)
Chloramphenicol	NA§	3	3	(17.6)	NA§	25	25	(9.2)
Erythromycin	0	0	0	—	0	0	0	—
Clindamycin	0	0	0	—	0	0	0	—
Rifampicin	0	0	0	—	0	0	0	—
H. influenzae	n=8 isolates				n=73 isolates			
Ampicillin¶	0	0	0	—	0	1	1	(1.4)
Cotrimoxazole†	0	0	0	—	2	7	9	(12.3)
Chloramphenicol	0	0	0	—	0	0	0	—

*Based on standards of the 1994 National Committee for Clinical Laboratory Standards (*3*).
†Trimethoprim-sulfamethoxazole.
§Not applicable (chloramphenicol has no intermediate resistance category).
¶Resistance is the same for amoxicillin.

of the 35 inpatients yielded 17 SP isolates and eight HI isolates. The study was conducted during the dry season and the period of peak pneumonia incidence in CAR.

Among outpatients, resistance rates for NP cultures were relatively low for ampicillin, penicillin, and cotrimoxazole (Table 1): 8.8% of SP isolates were resistant to penicillin, and 6.3% were resistant to cotrimoxazole. Among HI isolates, 1.4% were resistant to ampicillin, and 12.3% were resistant to cotrimoxazole; no beta-lactamase were resistant to chloramphenicol, and no HI isolates were resistant to chloramphenicol. Although SP resistance to tetracycline was high (42.3%), this antibiotic has not been recommended for children, and no recent tetracycline use was documented in this survey population. SP resistance was not documented for erythromycin, rifampicin, or clindamycin—drugs used infrequently in Central African children.

SP resistance rates to penicillin, cotrimoxazole, tetracycline, and chloramphenicol were higher among inpatients compared with outpatients (Table 1). In contrast, none of the HI isolates cultured from inpatients was resistant to ampicillin, cotrimoxazole, or chloramphenicol (Table 1).

Reported by: J Ndoyo, MD, RM Siopathis, MD, Ministry of Population and Public Health, Central African Republic. KP Klugman, MD, A Wasas, MT, Medical Research Council and Univ of the Witwatersrand Pneumococcal Diseases Research Unit, South African Institute for Medical Research, Johannesburg, South Africa. Childhood and Respiratory Diseases Br, Div of Bacterial and Mycotic Diseases; International Child Survival and Emerging Infections Program Support Activity, Div of Parasitic Diseases, National Center for Infectious Diseases, CDC.

Editorial Note: To reduce ARI deaths among children, the World Health Organization (WHO) recommends that children aged <5 years with clinical signs of pneumonia be treated empirically with an antibiotic that has activity against SP and HI (*1*). Antibiotic

S. pneumoniae *and* H. influenzae — *Continued*

resistance may decrease the clinical effectiveness of antibiotics used to treat pneumonia. The occurrence of SP and HI antibiotic resistance is a particular challenge for developing countries that can only afford inexpensive antibiotics (e.g., amoxicillin and cotrimoxazole).

The full extent of antibiotic resistance among developing countries in Africa is unknown. Previous reports suggest wide variation; however, differences in study design and laboratory methods make comparisons difficult (*4–7*). To standardize surveillance of SP and HI resistance, WHO and CDC have developed a manual that provides details on conducting resistance surveys similar to that described in this report (*8*).

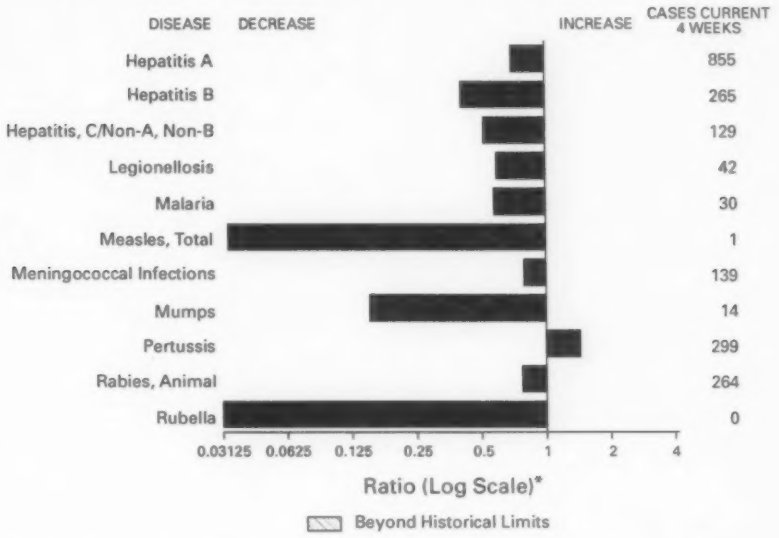
Because the clinical impact of antibiotic resistance on the treatment of children with pneumonia is not well understood, it is not clear when a national ARI program should change antibiotic recommendations in response to increasing resistance. In CAR, resistance rates were generally low and similar for cotrimoxazole and penicillin/amoxicillin; consequently, MOPHP recommended the use of cotrimoxazole as the first-line treatment for children with pneumonia because of its lower cost, twice-daily dosage, and antimalarial effect. In countries with severely limited budgets for health care and in which resistance to the recommended antibiotic is high, the costs and benefits of replacing a less expensive first-line antibiotic with a more expensive antibiotic to which respiratory pathogens have less resistance must be critically assessed.

Worldwide public health efforts should focus on improving surveillance, developing guidelines for the practical application of surveillance data, advocating policies for the rational use of antibiotics, and ensuring that children in need of antibiotic treatment for pneumonia are treated promptly and correctly. The impact of antibiotic resistance could be minimized by the testing and use of newly developed conjugate vaccines against *H. influenzae* type b and pneumococcal disease.

References

1. Mastro T, Nomani N, Ishaq Z, et al. Use of nasopharyngeal isolates of *Streptococcus pneumoniae* and *Haemophilus influenzae* from children in Pakistan for surveillance for antimicrobial resistance. Pediatr Infect Dis J 1993;12:824–30.
2. World Health Organization. Technical bases for the WHO recommendations on the management of pneumonia in children at first-level facilities. Geneva, Switzerland: World Health Organization, Programme for the Control of Acute Respiratory Infections, 1991; publication no. WHO/ARI/91.20.
3. National Committee for Clinical Laboratory Standards. Performance standards for antimicrobial susceptibility testing. Villanova, Pennsylvania: National Committee for Clinical Laboratory Standards, 1994; document no. M100-S5.
4. Bogaerts J, Lepage P, Taelman H, et al. Antimicrobial susceptibility and serotype distribution of *Streptococcus pneumoniae* from Rwanda, 1984–1990. J Infect 1993;27:157–68.
5. Klugman K. Pneumococcal resistance to antibiotics. Clin Microbiol Rev 1990;3:171–96.
6. Bijlmer HA, van Alphen L, Greenwood BM, Geelen-van den Broek L, Valkenburg HA, Dankert J. Antibiotic susceptibility of invasive and non-invasive isolates of *Haemophilus influenzae* from the Gambia, West Africa. J Antimicrob Chemother 1994;34:275–80.
7. Weinberg GA, Spitzer ED, Murray PR, et al. Antimicrobial susceptibility patterns of *Haemophilus* isolates from children in eleven developing nations. Bull World Health Organ 1990;68:179–84.
8. World Health Organization. Draft version of the manual for the national surveillance of antimicrobial resistance of *S. pneumoniae* and *H. influenzae*: epidemiological and microbiological methods. Geneva, Switzerland: World Health Organization, Program for the Control of Acute Respiratory Infections, August 1994.

FIGURE I. Selected notifiable disease reports, comparison of provisional 4-week totals ending January 18, 1997, with historical data — United States



*Ratio of current 4-week total to mean of 15 4-week totals (from previous, comparable, and subsequent 4-week periods for the past 5 years). The point where the hatched area begins is based on the mean and two standard deviations of these 4-week totals.

TABLE I. Summary — provisional cases of selected notifiable diseases, United States, cumulative, week ending January 18, 1997 (3rd Week)

	Cum. 1997		Cum. 1997
Anthrax	-	Plague	-
Brucellosis	1	Poliomyelitis, paralytic	-
Cholera	-	Psittacosis	-
Congenital rubella syndrome	-	Rabies, human	-
Cryptosporidiosis*	28	Rocky Mountain spotted fever (RMSF)	3
Diphtheria	-	Streptococcal disease, invasive Group A	10
Encephalitis: California*	-	Streptococcal toxic-shock syndrome*	2
eastern equine*	-	Syphilis, congenital†	-
St. Louis*	-	Tetanus	1
western equine*	-	Toxic-shock syndrome	2
Hansen Disease	4	Trichinosis	2
Hantavirus pulmonary syndrome*†	-	Typhoid fever	4
Hemolytic uremic syndrome, post-diarrheal*	-	Yellow fever	-
HIV infection, pediatric*§	-		

-no reported cases
*Not notifiable in all states.
†Updated weekly from reports to the Division of Viral and Rickettsial Diseases, National Center for Infectious Diseases (NCID).
§Updated monthly to the Division of HIV/AIDS Prevention, National Center for HIV, STD, and TB Prevention (NCHSTP), last update December 24, 1996.
¶Updated quarterly from reports to the Division of STD Prevention, NCHSTP.

TABLE II. Provisional cases of selected notifiable diseases, United States, weeks ending January 18, 1997, and January 20, 1996 (3rd Week)

Reporting Area	AIDS*		Chlamydia		*Escherichia coli* O157:H7		Gonorrhea		Hepatitis C/NA,NB	
					NETSS†	PHLIS§				
	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1997	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996
UNITED STATES	-	1,800	9,564	12,844	36	-	8,614	16,291	72	122
NEW ENGLAND	-	2	698	842	1	-	277	437	-	-
Maine	-	-	-	-	-	-	-	2	-	-
N.H.	-	2	2	33	-	-	-	7	-	-
Vt.	-	-	10	27	-	-	1	9	-	-
Mass.	-	-	431	346	1	-	142	184	-	-
R.I.	-	-	91	100	-	-	33	32	-	-
Conn.	-	-	164	336	-	-	101	203	-	-
MID. ATLANTIC	-	811	414	269	-	-	202	1,366	-	4
Upstate N.Y.	-	12	N	N	-	-	-	-	-	3
N.Y. City	-	682	-	-	-	-	-	713	-	1
N.J.	-	114	312	269	-	-	191	197	-	-
Pa.	-	3	102	-	N	-	11	456	-	-
E.N. CENTRAL	-	120	1,705	3,934	3	-	1,599	3,634	32	16
Ohio	-	66	659	828	1	-	601	738	3	-
Ind.	-	-	269	-	1	-	220	499	1	-
Ill.	-	3	605	1,297	-	-	317	1,095	-	4
Mich.	-	36	153	1,202	1	-	434	964	28	12
Wis.	-	15	19	607	N	-	27	338	-	-
W.N. CENTRAL	-	72	630	1,303	8	-	321	833	2	1
Minn.	-	20	-	-	5	-	U	-	-	-
Iowa	-	-	-	-	3	-	-	-	2	-
Mo.	-	51	472	624	-	-	276	592	-	1
N. Dak.	-	-	37	37	-	-	2	-	-	-
S. Dak.	-	-	49	30	-	-	9	4	-	-
Nebr.	-	-	17	366	-	-	2	59	-	-
Kans.	-	1	55	246	-	-	32	178	-	-
S. ATLANTIC	-	87	2,123	1,638	3	-	3,792	6,345	7	4
Del.	-	-	-	-	-	-	69	86	-	-
Md.	-	65	258	123	-	-	652	863	3	-
D.C.	-	2	N	N	-	-	331	278	-	-
Va.	-	1	395	638	N	-	325	590	-	-
W. Va.	-	6	-	-	N	-	15	33	-	3
N.C.	-	1	-	-	2	-	986	722	3	-
S.C.	-	-	-	-	-	-	262	1,004	1	1
Ga.	-	8	590	-	1	-	539	1,984	U	-
Fla.	-	4	880	877	-	-	613	785	-	-
E.S. CENTRAL	-	46	862	1,255	2	-	1,041	1,606	9	29
Ky.	-	2	290	307	1	-	229	221	-	-
Tenn.	-	26	153	499	1	-	133	547	2	29
Ala.	-	-	419	445	-	-	679	735	1	-
Miss.	-	18	-	4	-	-	-	103	6	-
W.S. CENTRAL	-	39	666	323	1	-	749	509	1	26
Ark.	-	19	26	61	1	-	62	253	-	-
La.	-	18	332	-	-	-	402	60	1	1
Okla.	-	1	308	262	-	-	285	196	-	25
Tex.	-	1	-	-	-	-	-	-	-	-
MOUNTAIN	-	54	863	582	8	-	217	484	19	34
Mont.	-	2	22	-	-	-	4	1	2	2
Idaho	-	-	70	64	-	-	9	5	5	6
Wyo.	-	-	25	31	-	-	3	4	8	8
Colo.	-	-	-	-	5	-	-	116	2	4
N. Mex.	-	-	273	200	2	-	57	51	1	9
Ariz.	-	36	366	61	N	-	131	244	1	2
Utah	-	16	86	93	1	-	7	28	-	3
Nev.	-	-	21	133	-	-	6	35	-	-
PACIFIC	-	569	1,603	2,698	10	-	416	1,077	2	8
Wash.	-	64	462	416	-	-	122	149	-	-
Oreg.	-	28	-	247	2	-	-	9	1	2
Calif.	-	470	1,043	1,980	8	-	258	866	-	5
Alaska	-	-	63	19	-	-	30	32	-	1
Hawaii	-	7	35	36	N	-	6	21	1	-
Guam	-	-	-	14	N	-	-	11	-	-
P.R.	-	-	N	N	-	U	30	-	-	5
V.I.	-	-	N	N	N	U	-	-	-	-
Amer. Samoa	-	-	-	-	N	U	-	-	-	-
C.N.M.I.	-	-	N	N	N	U	-	3	-	-

N: Not notifiable U: Unavailable -: no reported cases C.N.M.I.: Commonwealth of Northern Mariana Islands

*Updated monthly to the Division of HIV/AIDS Prevention, National Center for HIV, STD, and TB Prevention, last update December 24, 1996.
†National Electronic Telecommunications System for Surveillance.
§Public Health Laboratory Information System.

TABLE II. (Cont'd.) Provisional cases of selected notifiable diseases, United States, weeks ending January 18, 1997, and January 20, 1996 (3rd Week)

Reporting Area	Legionellosis		Lyme Disease		Malaria		Syphilis (Primary & Secondary)		Tuberculosis		Rabies, Animal
	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997
UNITED STATES	27	37	31	78	33	41	255	557	283	493	209
NEW ENGLAND	1	1	2	-	-	2	6	12	6	9	25
Maine	-	-	-	-	-	-	-	-	-	1	1
N.H.	-	-	-	-	-	-	-	-	-	-	-
Vt.	1	-	1	-	-	-	-	-	-	-	6
Mass.	-	-	-	-	-	2	2	4	1	-	2
R.I.	-	1	1	-	-	-	-	-	-	4	-
Conn.	N	N	-	-	-	-	4	8	5	4	16
MID. ATLANTIC	1	7	8	64	2	9	1	9	11	15	52
Upstate N.Y.	-	-	-	-	-	-	-	-	-	-	49
N.Y. City	-	-	1	11	1	2	-	5	-	3	-
N.J.	1	3	7	22	1	7	1	-	-	-	3
Pa.	-	4	-	31	-	-	-	4	11	12	-
E.N. CENTRAL	15	14	2	1	2	6	20	119	52	123	-
Ohio	8	4	1	1	-	-	11	48	31	8	-
Ind.	3	3	1	-	-	-	3	22	3	7	-
Ill.	-	1	-	-	-	2	6	32	18	108	-
Mich.	4	6	-	-	2	3	-	-	-	-	-
Wis.	-	-	U	U	-	1	-	17	-	-	-
W.N. CENTRAL	-	1	-	1	-	1	5	24	3	6	21
Minn.	-	-	-	-	-	-	-	-	3	1	2
Iowa	-	-	-	-	-	-	-	-	-	3	16
Mo.	-	1	-	1	-	1	5	16	-	1	-
N. Dak.	-	-	-	-	-	-	-	-	-	-	3
S. Dak.	-	-	-	-	-	-	-	-	-	-	-
Nebr.	-	-	-	-	-	-	-	3	-	-	-
Kans.	-	-	-	-	-	-	-	5	-	1	-
S. ATLANTIC	5	3	10	9	4	7	119	144	25	25	103
Del.	-	-	-	2	1	2	-	2	-	2	2
Md.	4	1	9	7	1	2	33	6	4	-	25
D.C.	1	-	-	-	-	1	6	6	5	-	1
Va.	-	-	-	-	-	1	14	31	-	-	-
W. Va.	-	1	-	-	-	-	-	-	1	4	1
N.C.	-	1	1	-	1	-	35	34	-	11	58
S.C.	-	-	-	-	1	-	-	9	15	8	-
Ga.	-	-	-	-	-	-	15	46	-	-	10
Fla.	-	-	-	-	-	1	16	10	-	-	6
E.S. CENTRAL	2	6	6	3	-	-	56	205	12	38	4
Ky.	-	3	-	-	-	-	10	16	-	2	2
Tenn.	-	1	-	3	-	-	11	50	-	7	-
Ala.	1	-	-	-	-	-	35	37	12	14	2
Miss.	1	2	6	-	-	-	-	102	-	15	-
W.S. CENTRAL	-	-	-	-	-	-	42	19	-	9	3
Ark.	-	-	-	-	-	-	-	11	-	-	-
La.	-	-	-	-	-	-	31	8	-	-	-
Okla.	-	-	-	-	-	-	11	-	-	9	3
Tex.	-	-	-	-	-	-	-	-	-	-	-
MOUNTAIN	1	2	-	-	1	2	3	13	4	23	-
Mont.	-	-	-	-	1	-	-	-	-	-	-
Idaho	-	-	-	-	-	-	-	-	-	1	-
Wyo.	-	-	-	-	-	-	-	-	1	-	-
Colo.	1	-	-	-	-	1	-	3	1	12	-
N. Mex.	-	-	-	-	-	-	-	-	2	-	-
Ariz.	-	1	-	-	-	-	3	8	-	10	-
Utah	-	-	-	-	-	1	-	-	-	-	-
Nev.	-	1	-	-	-	-	-	2	-	-	-
PACIFIC	2	3	3	-	24	14	3	12	170	245	1
Wash.	-	-	-	-	-	-	-	-	3	10	-
Oreg.	-	-	1	-	2	2	-	1	-	4	-
Calif.	2	3	2	-	22	12	3	11	146	218	1
Alaska	-	-	-	-	-	-	-	-	5	7	-
Hawaii	-	-	-	-	-	-	-	-	16	6	-
Guam	-	-	-	-	-	-	-	1	-	-	-
P.R.	-	-	-	-	-	-	8	-	-	-	-
V.I.	-	-	-	-	-	-	-	-	-	-	-
Amer. Samoa	-	-	-	-	-	-	-	-	-	-	-
C.N.M.I.	-	-	-	-	-	-	-	-	-	-	-

N: Not notifiable U: Unavailable -: no reported cases

TABLE III. Provisional cases of selected notifiable diseases preventable by vaccination, United States, weeks ending January 18, 1997, and January 20, 1996 (3rd Week)

Reporting Area	*H. influenzae*, invasive		Hepatitis (Viral), by type				Measles (Rubeola)					
			A		B		Indigenous		Imported[†]		Total	
	Cum. 1997*	Cum. 1996	Cum. 1997	Cum. 1996	Cum. 1997	Cum. 1996	1997	Cum. 1997	1997	Cum. 1997	Cum. 1997	Cum. 1996
UNITED STATES	41	62	721	1,119	217	335	-	-	-	-	-	1
NEW ENGLAND	2	3	15	9	1	10	-	-	-	-	-	-
Maine	-	-	1	1	-	-	-	-	-	-	-	-
N.H.	-	3	-	1	-	-	-	-	-	-	-	-
Vt.	-	-	2	-	-	-	-	-	-	-	-	-
Mass.	2	-	3	1	1	-	-	-	-	-	-	-
R.I.	-	-	-	2	-	1	-	-	-	-	-	-
Conn.	-	-	9	4	-	9	-	-	-	-	-	-
MID. ATLANTIC	7	7	42	35	30	28	-	-	-	-	-	-
Upstate N.Y.	-	-	-	2	-	3	-	-	-	-	-	-
N.Y. City	3	-	20	7	14	8	-	-	-	-	-	-
N.J.	4	5	22	16	16	13	-	-	-	-	-	-
Pa.	-	2	-	10	-	4	-	-	-	-	-	-
E.N. CENTRAL	3	9	65	129	28	62	-	-	-	-	-	-
Ohio	3	8	26	56	4	6	-	-	-	-	-	-
Ind.	-	-	14	3	2	3	-	-	-	-	-	-
Ill.	-	1	-	36	-	26	-	-	-	-	-	-
Mich.	-	-	25	17	22	18	-	-	-	-	-	-
Wis.	-	-	-	17	-	9	U	-	U	-	-	-
W.N. CENTRAL	-	5	10	88	11	22	-	-	-	-	-	-
Minn.	-	-	-	-	-	1	-	-	-	-	-	-
Iowa	-	3	9	22	10	5	-	-	-	-	-	-
Mo.	-	2	-	42	-	12	-	-	-	-	-	-
N. Dak.	-	-	-	-	-	-	-	-	-	-	-	-
S. Dak.	-	-	1	4	-	-	-	-	-	-	-	-
Nebr.	-	-	-	16	1	1	-	-	-	-	-	-
Kans.	-	-	-	4	-	3	U	-	U	-	-	-
S. ATLANTIC	12	3	38	22	20	47	-	-	-	-	-	-
Del.	-	-	4	1	1	-	-	-	-	-	-	-
Md.	4	-	23	10	8	16	-	-	-	-	-	-
D.C.	2	-	1	-	1	1	-	-	-	-	-	-
Va.	-	-	-	-	-	1	-	-	-	-	-	-
W. Va.	1	-	1	1	1	3	-	-	-	-	-	-
N.C.	4	1	6	3	7	22	-	-	-	-	-	-
S.C.	-	-	1	4	2	3	-	-	-	-	-	-
Ga.	-	2	1	-	-	-	-	-	-	-	-	-
Fla.	1	-	1	3	-	1	-	-	-	-	-	-
E.S. CENTRAL	1	2	20	67	13	41	-	-	-	-	-	-
Ky.	1	-	-	4	-	4	-	-	-	-	-	-
Tenn.	-	1	-	36	-	35	-	-	-	-	-	-
Ala.	-	1	5	4	1	2	-	-	-	-	-	-
Miss.	-	-	15	23	12	U	-	-	-	-	-	-
W.S. CENTRAL	2	3	49	127	3	7	-	-	-	-	-	-
Ark.	-	-	10	15	3	1	-	-	-	-	-	-
La.	-	-	-	-	-	1	-	-	-	-	-	-
Okla.	2	3	37	103	-	5	-	-	-	-	-	-
Tex.	-	-	2	9	-	-	-	-	-	-	-	-
MOUNTAIN	1	6	150	177	44	45	-	-	-	-	-	-
Mont.	-	-	5	5	-	-	-	-	-	-	-	-
Idaho	-	1	14	25	-	3	-	-	-	-	-	-
Wyo.	-	-	2	-	1	-	-	-	-	-	-	-
Colo.	1	1	31	12	11	8	-	-	-	-	-	-
N. Mex.	-	2	13	38	22	19	-	-	-	-	-	-
Ariz.	-	1	34	28	7	4	-	-	-	-	-	-
Utah	-	-	45	47	2	7	-	-	-	-	-	-
Nev.	-	1	6	22	1	4	-	-	-	-	-	-
PACIFIC	13	24	332	465	67	73	-	-	-	-	-	1
Wash.	-	-	-	2	-	-	-	-	-	-	-	-
Oreg.	3	2	31	106	12	7	-	-	-	-	-	-
Calif.	8	22	291	356	54	66	-	-	-	-	-	-
Alaska	-	-	2	-	-	-	-	-	-	-	-	-
Hawaii	2	-	8	1	1	-	-	-	-	-	-	1
Guam	-	-	-	1	-	-	U	-	U	-	-	-
P.R.	-	-	2	1	1	4	-	-	-	-	-	-
V.I.	-	-	-	-	-	-	U	-	U	-	-	-
Amer. Samoa	-	-	-	-	-	-	U	-	U	-	-	-
C.N.M.I.	-	6	-	1	-	3	U	-	U	-	-	-

N: Not notifiable U: Unavailable -: no reported cases

*Of 6 cases among children aged <5 years, serotype was reported for 1 and of those, 0 were type b.

†For imported measles, cases include only those resulting from importation from other countries.

TABLE III. (Cont'd.) Provisional cases of selected notifiable diseases preventable by vaccination, United States, weeks ending January 18, 1997, and January 20, 1996 (3rd Week)

Reporting Area	Meningococcal Disease		Mumps			Pertussis			Rubella		
	Cum. 1997	Cum. 1996	1997	Cum. 1997	Cum. 1996	1997	Cum. 1997	Cum. 1996	1997	Cum. 1997	Cum. 1996
UNITED STATES	130	220	1	2	20	21	150	54	-	-	9
NEW ENGLAND	8	13	-	-	-	5	24	5	-	-	-
Maine	-	3	-	-	-	-	3	-	-	-	-
N.H.	-	-	-	-	-	-	3	-	-	-	-
Vt.	-	1	-	-	-	5	18	2	-	-	-
Mass.	5	2	-	-	-	-	-	3	-	-	-
R.I.	-	1	-	-	-	-	-	-	-	-	-
Conn.	3	6	-	-	-	-	-	-	-	-	-
MID. ATLANTIC	8	14	-	-	3	-	-	1	-	-	-
Upstate N.Y.	-	-	-	-	-	-	-	-	-	-	-
N.Y. City	4	4	-	-	-	-	-	-	-	-	-
N.J.	4	5	-	-	2	-	-	1	-	-	-
Pa.	-	5	-	-	1	-	-	-	-	-	-
E.N. CENTRAL	16	40	-	-	8	1	5	16	-	-	-
Ohio	11	19	-	-	5	-	-	7	-	-	-
Ind.	3	3	-	-	-	-	-	-	-	-	-
Ill.	-	12	-	-	1	-	-	1	-	-	-
Mich.	2	2	-	-	2	1	5	3	-	-	-
Wis.	-	4	U	-	-	U	-	5	U	-	-
W.N. CENTRAL	5	26	-	-	-	3	5	1	-	-	-
Minn.	-	-	-	-	-	-	-	-	-	-	-
Iowa	5	5	-	-	-	3	4	-	-	-	-
Mo.	-	14	-	-	-	-	-	1	-	-	-
N. Dak.	-	-	-	-	-	-	-	-	-	-	-
S. Dak.	-	1	-	-	-	-	1	-	-	-	-
Nebr.	-	3	-	-	-	-	-	-	-	-	-
Kans.	-	3	U	-	-	U	-	-	U	-	-
S. ATLANTIC	32	23	-	-	1	1	3	1	-	-	-
Del.	2	-	-	-	-	-	-	-	-	-	-
Md.	2	4	-	-	-	1	3	-	-	-	-
D.C.	1	2	-	-	-	-	-	-	-	-	-
Va.	-	-	-	-	-	-	-	-	-	-	-
W. Va.	1	-	-	-	-	-	-	-	-	-	-
N.C.	6	2	-	-	-	-	-	-	-	-	-
S.C.	12	7	-	-	1	-	-	-	-	-	-
Ga.	5	7	-	-	-	-	-	1	-	-	-
Fla.	3	1	-	-	-	-	-	-	-	-	-
E.S. CENTRAL	13	18	1	2	1	-	1	6	-	-	-
Ky.	-	4	-	-	-	-	-	5	-	-	-
Tenn.	-	4	-	-	-	-	-	-	-	-	-
Ala.	9	8	-	-	1	-	1	1	-	-	-
Miss.	4	2	1	2	-	-	-	-	-	-	N
W.S. CENTRAL	3	14	-	-	1	-	-	1	-	-	-
Ark.	2	3	-	-	-	-	-	1	-	-	-
La.	-	3	-	-	1	-	-	-	-	-	-
Okla.	1	2	-	-	-	-	-	-	-	-	-
Tex.	-	6	-	-	-	-	-	-	-	-	-
MOUNTAIN	5	16	-	-	1	9	85	12	-	-	-
Mont.	1	-	-	-	-	-	-	-	-	-	-
Idaho	-	1	-	-	-	3	70	-	-	-	-
Wyo.	-	-	-	-	-	-	1	-	-	-	-
Colo.	-	2	-	-	-	3	8	-	-	-	-
N. Mex.	2	5	N	N	N	3	3	4	-	-	-
Ariz.	1	5	-	-	-	-	3	-	-	-	-
Utah	1	1	-	-	-	-	-	-	-	-	-
Nev.	-	2	-	-	1	-	-	8	-	-	-
PACIFIC	40	56	-	-	5	2	27	11	-	-	9
Wash.	-	-	-	-	-	-	-	-	-	-	-
Oreg.	19	14	-	-	-	-	2	10	-	-	-
Calif.	21	41	-	-	4	2	25	-	-	-	9
Alaska	-	1	-	-	-	-	-	-	-	-	-
Hawaii	-	-	-	-	1	-	-	1	-	-	-
Guam	-	1	U	-	1	U	-	-	U	-	-
P.R.	-	-	-	-	-	-	-	-	-	-	-
V.I.	-	-	U	-	-	U	-	-	U	-	-
Amer. Samoa	-	-	U	-	-	U	-	-	U	-	-
C.N.M.I.	-	-	U	-	-	U	-	-	U	-	-

N: Not notifiable U: Unavailable -: no reported cases

TABLE IV. Deaths in 122 U.S. cities,* week ending January 18, 1997 (3rd Week)

Reporting Area	All Causes, By Age (Years)						P&I† Total
	All Ages	>65	45-64	25-44	1-24	<1	
NEW ENGLAND	598	442	90	40	12	14	57
Boston, Mass.	168	114	29	16	5	4	22
Bridgeport, Conn.	40	29	9	1	1	-	5
Cambridge, Mass.	16	12	3	1	-	-	1
Fall River, Mass.	28	20	5	2	-	1	-
Hartford, Conn.	65	46	7	6	1	5	1
Lowell, Mass.	33	28	4	1	-	-	5
Lynn, Mass.	15	11	3	1	-	-	1
New Bedford, Mass.	20	19	1	-	-	-	1
New Haven, Conn.	36	24	5	3	2	2	2
Providence, R.I.	U	U	U	U	U	U	U
Somerville, Mass.	12	11	-	1	-	-	-
Springfield, Mass.	65	49	7	6	1	2	3
Waterbury, Conn.	50	38	9	1	2	-	6
Worcester, Mass.	50	41	8	1	-	-	10
MID. ATLANTIC	2,739	1,924	509	207	56	43	194
Albany, N.Y.	51	39	7	3	-	2	6
Allentown, Pa.	19	15	4	-	-	-	1
Buffalo, N.Y.	88	65	15	4	3	1	4
Camden, N.J.	51	27	16	5	1	2	5
Elizabeth, N.J.	22	18	2	2	-	-	1
Erie, Pa.§	66	47	14	3	2	-	7
Jersey City, N.J.	54	41	3	8	1	1	5
New York City, N.Y.	1,391	963	261	122	24	21	78
Newark, N.J.	51	24	14	7	6	-	7
Paterson, N.J.	27	20	3	2	-	2	1
Philadelphia, Pa.	400	254	100	27	14	5	27
Pittsburgh, Pa.§	104	75	19	7	-	3	11
Reading, Pa.	11	10	1	-	-	-	4
Rochester, N.Y.	150	119	21	3	2	5	14
Schenectady, N.Y.	30	23	6	1	-	-	1
Scranton, Pa.§	45	39	5	1	-	-	2
Syracuse, N.Y.	96	80	10	5	1	-	11
Trenton, N.J.	31	21	5	3	1	1	4
Utica, N.Y.	25	22	2	1	-	-	3
Yonkers, N.Y.	27	22	1	3	1	-	2
E.N. CENTRAL	2,649	1,876	472	179	60	62	239
Akron, Ohio	87	70	11	1	1	4	1
Canton, Ohio	46	39	6	-	-	1	11
Chicago, Ill.	464	282	94	48	21	19	36
Cincinnati, Ohio	223	161	36	17	5	4	31
Cleveland, Ohio	188	131	36	15	4	2	4
Columbus, Ohio	227	165	43	14	3	2	35
Dayton, Ohio	172	141	22	4	3	2	23
Detroit, Mich.	252	151	54	33	5	9	16
Evansville, Ind.	54	38	13	2	-	1	2
Fort Wayne, Ind.	94	78	13	2	1	-	12
Gary, Ind.	U	U	U	U	U	U	U
Grand Rapids, Mich.	78	57	11	7	2	1	16
Indianapolis, Ind.	223	154	42	14	5	8	-
Lansing, Mich.	33	20	8	3	2	-	8
Milwaukee, Wis.	140	100	25	10	3	2	14
Peoria, Ill.	33	28	4	-	-	1	4
Rockford, Ill.	47	39	6	1	1	-	4
South Bend, Ind.	53	42	6	3	1	1	8
Toledo, Ohio	156	118	30	3	3	2	12
Youngstown, Ohio	79	62	12	2	-	3	2
W.N. CENTRAL	918	689	128	55	13	18	93
Des Moines, Iowa	92	77	12	3	-	-	10
Duluth, Minn.	39	33	3	1	1	1	5
Kansas City, Kans.	26	18	6	1	1	-	1
Kansas City, Mo.	101	65	14	4	2	1	9
Lincoln, Nebr.	49	38	6	4	1	-	7
Minneapolis, Minn.	228	175	27	16	3	7	23
Omaha, Nebr.	142	101	24	10	3	4	14
St. Louis, Mo.	114	78	20	11	2	3	6
St. Paul, Minn.	80	64	12	3	-	1	11
Wichita, Kans.	47	40	4	2	-	1	7
S. ATLANTIC	1,599	1,063	318	150	36	26	113
Atlanta, Ga.	246	146	53	33	10	4	11
Baltimore, Md.	273	186	41	32	9	5	35
Charlotte, N.C.	100	71	16	10	3	-	5
Jacksonville, Fla.	162	107	37	13	2	2	12
Miami, Fla.	91	66	14	11	-	-	-
Norfolk, Va.	75	47	20	5	2	1	5
Richmond, Va.	U	U	U	U	U	U	U
Savannah, Ga.	88	63	17	5	-	3	9
St. Petersburg, Fla.	66	46	14	3	1	2	4
Tampa, Fla.	274	192	53	17	2	8	27
Washington, D.C.	204	124	50	22	7	1	5
Wilmington, Del.	20	15	3	2	-	-	-
E.S. CENTRAL	1,031	728	180	79	27	16	85
Birmingham, Ala.	91	60	17	6	3	4	3
Chattanooga, Tenn.	120	87	17	11	4	1	9
Knoxville, Tenn.	91	70	15	4	1	1	10
Lexington, Ky.	51	34	14	3	-	-	7
Memphis, Tenn.	215	145	41	17	9	3	16
Mobile, Ala.	139	87	31	14	4	3	4
Montgomery, Ala.	77	58	7	7	3	2	7
Nashville, Tenn.	247	187	38	17	3	2	29
W.S. CENTRAL	1,616	1,076	315	128	54	42	136
Austin, Tex.	73	46	15	4	5	3	9
Baton Rouge, La.	105	68	20	10	5	2	2
Corpus Christi, Tex.	81	60	14	7	-	-	9
Dallas, Tex.	220	133	50	25	9	3	9
El Paso, Tex.	178	121	36	10	6	4	19
Ft. Worth, Tex.	66	45	11	2	3	5	2
Houston, Tex.	343	212	81	33	8	9	33
Little Rock, Ark.	U	U	U	U	U	U	U
New Orleans, La.	67	40	11	8	5	3	-
San Antonio, Tex.	287	200	49	21	8	9	29
Shreveport, La.	79	61	10	3	3	2	8
Tulsa, Okla.	117	90	18	5	2	2	16
MOUNTAIN	1,188	861	199	81	33	14	136
Albuquerque, N.M.	122	77	26	14	5	-	7
Boise, Idaho	41	32	5	4	-	-	3
Colo. Springs, Colo.	52	38	5	7	2	-	5
Denver, Colo.	126	97	18	8	-	3	12
Las Vegas, Nev.	260	173	59	20	6	2	27
Ogden, Utah	35	32	2	-	-	1	7
Phoenix, Ariz.	203	134	39	18	6	4	23
Pueblo, Colo.	27	22	3	2	-	-	-
Salt Lake City, Utah	135	98	20	4	9	4	19
Tucson, Ariz.	187	158	22	4	3	-	33
PACIFIC	1,869	1,362	293	133	48	33	167
Berkeley, Calif.	18	14	2	1	-	1	-
Fresno, Calif.	59	45	8	3	1	2	6
Glendale, Calif.	16	13	2	-	1	-	1
Honolulu, Hawaii	77	57	14	4	-	2	5
Long Beach, Calif.	71	54	7	8	1	1	12
Los Angeles, Calif.	395	285	57	29	18	6	16
Pasadena, Calif.	39	29	4	2	-	4	5
Portland, Oreg.	218	168	32	9	6	3	16
Sacramento, Calif.	U	U	U	U	U	U	U
San Diego, Calif.	182	111	42	21	6	2	22
San Francisco, Calif.	160	107	35	16	1	1	25
San Jose, Calif.	195	147	30	7	7	4	25
Santa Cruz, Calif.	36	27	5	3	1	-	2
Seattle, Wash.	252	189	32	22	5	4	10
Spokane, Wash.	73	53	14	3	1	2	14
Tacoma, Wash.	78	63	9	5	-	1	8
TOTAL	14,207¶	10,021	2,504	1,055	339	268	1,220

U: Unavailable -: no reported cases

*Mortality data in this table are voluntarily reported from 122 cities in the United States, most of which have populations of 100,000 or more. A death is reported by the place of its occurrence and by the week that the death certificate was filed. Fetal deaths are not included.

†Pneumonia and influenza.

§Because of changes in reporting methods in these 3 Pennsylvania cities, these numbers are partial counts for the current week. Complete counts will be available in 4 to 6 weeks.

¶Total includes unknown ages.

Contributors to the Production of the *MMWR* (Weekly)

Weekly Notifiable Disease Morbidity Data and 122 Cities Mortality Data

Denise Koo, M.D., M.P.H.
Deborah A. Adams
Timothy M. Copeland
Patsy A. Hall
Carol M. Knowles
Sarah H. Landis
Myra A. Montalbano

Desktop Publishing and Graphics Support

Morie M. Higgins
Peter M. Jenkins

The *Morbidity and Mortality Weekly Report (MMWR)* Series is prepared by the Centers for Disease Control and Prevention (CDC) and is available free of charge in electronic format and on a paid subscription basis for paper copy. To receive an electronic copy on Friday of each week, send an e-mail message to *lists@list.cdc.gov*. The body content should read *subscribe mmwr-toc*. Electronic copy also is available from CDC's World-Wide Web server at *http://www.cdc.gov/* or from CDC's file transfer protocol server at *ftp.cdc.gov*. To subscribe for paper copy, contact Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone (202) 512-1800.

Data in the weekly *MMWR* are provisional, based on weekly reports to CDC by state health departments. The reporting week concludes at close of business on Friday; compiled data on a national basis are officially released to the public on the following Friday. Address inquiries about the *MMWR* Series, including material to be considered for publication, to: Editor, *MMWR* Series, Mailstop C-08, CDC, 1600 Clifton Rd., N.E., Atlanta, GA 30333; telephone (404) 332-4555.

☆U.S. Government Printing Office: 1997-532-228/47053 Region IV

DEPARTMENT OF
HEALTH AND HUMAN SERVICES
Public Health Service
Centers for Disease Control
and Prevention (CDC)
Atlanta, Georgia 30333

Official Business
Penalty for Private Use $300
Address Correction Requested

FIRST-CLASS MAIL
POSTAGE & FEES PAID
PHS/CDC
Permit No. G-284